APHASIA IN STROKE -PATTERN AND PROGNOSIS - A PROSPECTIVE STUDY

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CERTIFICATE

This is to certify that **Dr.V.RAJASRI** is a bonafide student of the Institute of Internal Medicine, Madras Medical College, Chennai and this study titled **"APHASIA IN STROKE- PATTERN AND PROGNOSIS -A PROSPECTIVE STUDY"** is the original work done by her for her dissertation towards partial fulfillment of M.D.Degree 2003-2006.

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DECLARATION

I solemnly declare that the dissertation titled "**APHASIA IN STROKE-PATTERN AND PROGNOSIS-A PROSPECTIVE STUDY**" was done by me at Government General Hospital, Madras Medical College, Chennai between 2003-2005 under the guidance and supervision of **Prof.V.Sundaravadivelu** (Additional Professor of Medicine, MMC, Chennai)

The dissertation is submitted to the Tamilnadu Dr M.G.R Medical University towards partial fulfillment of requirements for the award of M.D.Degree –Branch I -General Medicine.

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INTRODUCTION

Stroke by definition is a syndrome of rapidly developing clinical signs of focal (or global) disturbance of cerebral function, lasting for more than 24 hours or leading to death, with no apparent cause other than vascular origin [1]. Stroke is the leading cause of disability and death all around the world. In India, community surveys have shown a crude prevalence rate of “hemiplegia” in the range of 200 per 100,000 persons, constituting nearly 1.5% of all hospital admissions, 4.5% of all medical and 20% of all neurological admissions [2].

The vascular pathologic processes that lead to stroke may be thrombosis, embolism, dissection or rupture of the vessel. These in turn can lead to two types of parenchymal changes in the brain- ischaemia with or without infarction or a haemorrhage. Atherosclerotic vascular disease is the most common cause for stroke all round the world, but other causes like arteritis (due to tuberculosis etc) and cardioembolic stroke contribute significantly especially in India [2]. The most important risk factors for stroke are hypertension, heart disease, atrial fibrillation, diabetes mellitus, cigarette smoking and hyperlipidemia.

Among the stroke survivors about one-third require assistance in various activities in daily living. Dependent living is related mainly to motor and cognitive deficits [3]. Aphasia, the loss or impairment of language caused by brain damage, is one of the most devastating cognitive impairments of stroke. Aphasia is observed with a frequency ranging from 21% to 38% in acute patients, [3-6] and stroke accounts for most of the new cases of aphasia seen in neurological practice. The presence of aphasia is an index of poor prognosis, with more severe motor, cognitive, and social disability [3,5] and higher mortality [5,6] Finally, aphasia outcome remains poor - 32% to 50% of aphasics still suffer from aphasia 6 months after stroke [5,6]. Therefore it is important to evaluate and treat post stroke aphasia.

The characteristics of vascular aphasias remain partly unknown for varied reasons [7]. Characteristics of aphasia in patients admitted to acute stroke units have been reported very infrequently in literature. Studies focusing on vascular aphasia have included either selected samples with extensive evaluation of language [3,8] or large series of consecutive patients examined with shortened testing [4,5,6]. Although the latter studies provide important findings, they usually focus on oral expression and do not determine the type of language disorder. This leads to confounding of very different disorders such as dysarthria and aphasia, or Broca’s and global

aphasias, which have different severities and outcomes. Factors determining the occurrence and severity of post stroke aphasia remain a subject of controversy. Lesion location has long been regarded as the major determinant of aphasia characteristics. However, several CT-based studies have shown that an unexpectedly large proportion of aphasias deviate from classic clinical-anatomic correlations [9, 10]. This suggests that other factors such as age, sex and initial severity of stroke influence clinical outcome.

The goal of this study is to determine characteristics of patients with aphasia at the acute stage of stoke ,the influence of age ,sex and stoke severity on aphasia type and the evolution of the aphasia with time.

AIM OF STUDY

The aim of this study is to evaluate aphasia characteristics at the time of presentation in patients with acute stroke and evaluate the changes in language function after 6 months.

1. To study the types of aphasia and the differences in age, sex characteristics of patients with different types of aphasia in acute stroke patients (within 2 weeks) admitted in Government General Hospital, Chennai.

2. To study the pattern of recovery of aphasia in patients after 6 months of initial presentation and to determine the factors which affect the prognosis of the aphasia due to stroke.

REVIEW OF LITERATURE

Speech and language functions are of fundamental human significance, both in social interaction and in private intellectual life. When they are disturbed as a consequence of brain disease, like stroke the resultant functional loss is profound. Language function involves the comprehension, formulation, and transmission of ideas and feelings by the use of conventionalized verbal symbols, sounds and gestures and their sequential ordering according to accepted rules of grammar. A derangement of language function is always a reflection of an abnormality of the brain and, usually of the dominant cerebral hemisphere. Historically, language was the first higher cortical function to be correlated with specific sites of brain damage.

Aphasia is defined as a disorder of language that is acquired secondary to brain damage[15]. This definition adapted from Alexander and Benson, separates aphasia from several related disorders. First aphasia is distinguished from congenital or developmental language disorders, called dysphasias. Second, aphasia is a disorder of language rather than speech. Speech is the articulation and phonation of language sounds. Aphasia is distinguished from motor speech disorders, which include dysarthria,

dysphonia, stuttering, and speech apraxia. Third, aphasia is distinguished from disorders of thought. Psychiatric disorders derange thought and alter the contents of speech without affecting its linguistic structure.

The functional supremacy of one cerebral is fundamental to language function. Approximately 90 to 95% of population is right handed. In right handed individuals aphasia is almost invariability related to left hemisphere lesions. Very rarely (less than 1%) of right handed individuals may develop aphasia after a right hemisphere lesion (crossed aphasia in dextrals). In a large series of left handed patients with aphasia, 60% had lesions confined to the left cerebral hemisphere (Goodglass)[16]. In left handed aphasic patients with right hemisphere lesion, the language disorder is often less severe and enduring than in right handers with comparable lesions in left hemisphere [17]. The latter finding suggests a bilateral albeit unequal representation of language functions in non-right handed individuals.

The language capacities of the non-dominant cerebral hemisphere have not been documented by careful anatomic studies. The observation of Levine and Mohr suggest that the non dominant hemisphere has only a limited ability to produce oral speech after extensive damage to the dominant hemisphere [18]. The right hemisphere does have an important

role in communicating emotions and feelings. These aspects of language are subsumed under the term prosody. Aprosodia is present in patients with lesions in the territory to the inferior division of the right middle cerebral artery.

Anatomy of the Language Functions

The conventional teaching, based on correlations between various disorders of language and damage to particular areas of the brain, postulates four main language areas, situated, in most persons, in the left cerebral hemisphere. The entire language zone that encompasses these areas is perisylvian, i.e., it borders the sylvian fissure. Two language areas are receptive and two are executive, i.e., concerned with the production (output) of language. The two receptive areas are closely related and embrace what has been referred to as the central language zone. One, sub serving the perception of spoken language, occupies the posterior-posterosuperior temporal area (the posterior portion of area 22) and Heschl's gyri (areas 41 and 42); the posterior part of area 22 in the planum temporale is referred to as Wernicke's area. A second area, subserving the perception of written language, occupies the angular gyrus (area 39) in the inferior parietal lobule, anterior to the visual receptive areas. The supramarginal gyrus, which lies

between these auditory and visual language "centers" and the inferior temporal region, just anterior to the visual association cortex, are probably part of this central language zone as well. Here are located the integrative centers for cross - modal visual and auditory language functions.

The main executive region, situated at the posterior end or the inferior frontal convolution (Brodmann areas 44 and 45), is referred to as Broca's area and is concerned with motor aspects of speech. Visually perceived words are given expression in writing through a fourth language area, the so-called Exner writing area in the posterior part of the second frontal convolution.

These sensory and motor areas are intricately connected with one another by a rich network of nerve fibers, one large bundle of which, the arcuate fasciculus, passes through the isthmus of the temporal lobe and around the posterior end of the sylvian fissure; other connections may traverse the external capsule of the lenticular nucleus (subcortical white matter of the insula). Many additional corticocortical connections and other fiber systems lead into the perisylvian zones and project from them to other parts of the brain. The visual receptive and somatosensory zones are integrated in the parietal lobe, and the auditory receptive zones, in the

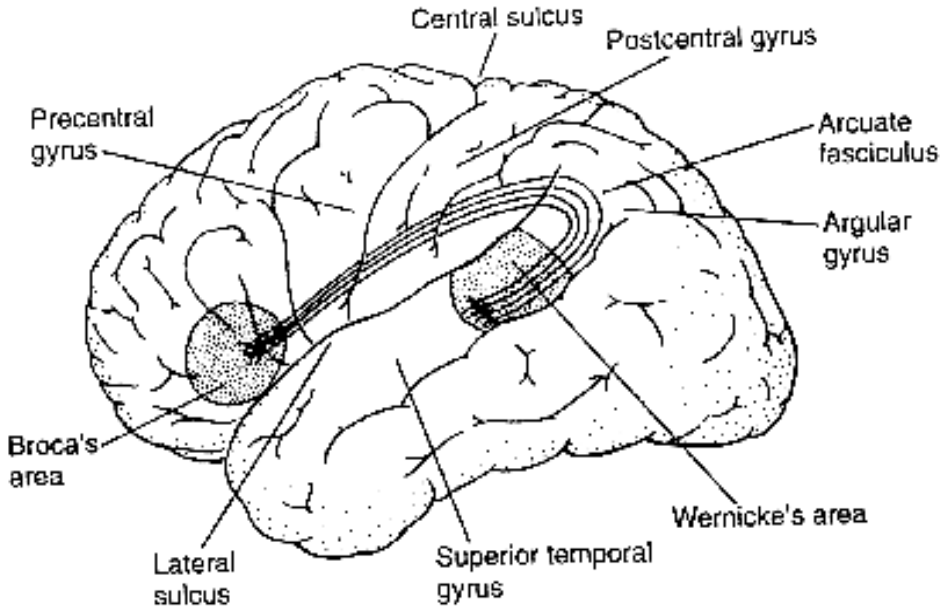
temporal lobe. Of special importance are the short association fibers that join Broca's area with the lower rolandic cortex, which, in turn, innervates the speech apparatus, i.e., the muscles of the lips, tongue, pharynx, and larynx. The Exner writing area is similarly integrated with the motor apparatus for the muscles of the hand. The perisylvian language areas are also connected with the striatum and thalamus and with corresponding areas in the nondominant cerebral hemisphere through the corpus callosum and anterior commissure.

There has been much difference of opinion concerning the cortical language areas, and objection has been made to calling them "centers", for they do not represent histologically circumscribed structures of constant function. Subcortical structures also play an increasingly recognized role in language function. Lesions of the dominant thalamus have been reported to cause fluent aphasia. The basal ganglia are involved in expressive speech.

Knowledge of the anatomy of language has come almost exclusively from the post mortem study of humans with focal brain diseases. Two major theories emerged from these studies. One subdivided the language zone into separate afferent (auditory and visual) receptive parts, connected by identifiable tracts to the executive (efferent-expressive) centers, Depending

on the exact anatomy of the lesions, a number of special syndromes are elicited. The other theory, favored a single language mechanism, roughly localized in the opercular, or perisylvian, region of the dominant cerebral hemisphere. The aphasia in any particular case was presumably due to the summation of damage to input or out put modalities consequent upon damage to this central language process.[36]

Despite the level of theoretical sophistication attempts to attempts to delineate the anatomy of speech and language using brain imaging techniques has been disappointing. Functional MRI has proved to be superior to computerized tomography but only the broadest rules of localization can be confirmed.



CLASSIFICATION OF APHASIA

As a consequence complexity and variability of aphasia, over the years a number of systems have been developed to classify various aphasia syndromes. Aphasia can be broadly divided as fluent and nonfluent (Benson, 1967) or as anterior and posterior (Goodglass and Kaplan, 1972) or as motor and sensory (originally proposed by Wernicke).

The Boston Aphasia Classification System, developed by the Speech Pathology Section of the Veterans Administration Hospital, Boston is a modification of Benson's Boston classification system[19]. According to this system, there are eight clinically recognizable aphasia syndromes, each characterized by a particular cluster of language signs and symptoms. They include

1. Broca's aphasia
2. Wernicke's aphasia
3. Conduction aphasia
4. Global aphasia
5. Transcortical motor aphasia
6. Transcortical sensory aphasia
7. Isolation aphasia
8. Anomic aphasia

Broca's Aphasia

In 1861, the French physician Paul Broca described two patients, establishing the aphasic syndrome that now bears his name. The speech pattern is nonfluent; on bedside examination, the patient speaks hesitantly, often producing the principal, meaning-containing nouns and verbs but omitting small grammatical words and morphemes. This pattern is called agrammatism or telegraphic speech. Patients with acute Broca's aphasia may be mute or may produce only single words, often with dysarthria and apraxia of speech. They make many phonemic errors, inconsistent from utterance to utterance, with substitution of phonemes usually differing only slightly from the correct target (e.g. for b). Naming is deficient, but the patient often manifests a "tip of the tongue" phenomenon, getting out the first letter of phoneme of the correct name. Paraphasic errors in naming are more often of the literal type than the verbal type. Auditory comprehension seems intact, but detailed testing usually reveals some deficiency, particularly in the comprehension of complex syntax. Repetition is hesitant in these patients, resembling their spontaneous speech.

Reading is often impaired despite relatively preserved auditory comprehension. Benson termed this reading difficulty of Broca's aphasics

the "third alexia," in distinction to the two classic types of alexia. Writing is virtually always deficient in Broca's aphasics. Most patients have a right hemiparesis, necessitating use of the nondominant, left hand for writing, but this left-handed writing is far more abnormal than the awkward renditions of a normal, right handed patient. Many patients can scrawl only a few letters.

Associated neurological deficits of Broca's aphasia include right hemiparesis, hemisensory loss, and apraxia of the oral apparatus and the nonparalyzed left limbs. Apraxia in response to motor commands in important to recognize because it may be mistaken for comprehension disturbance.

An important clinical feature of Broca's aphasia is its frequent association with depression. [40] Patients with Broca's aphasia are typically aware of and frustrated by their deficits. At times they become with drawn and refuse help or therapy. Usually, the depression lifts as the deficit recovers, although it may be a limiting factor in rehabilitation.

The lesions responsible for Broca's aphasia usually include the traditional Broca's area in the posterior part of the inferior frontal gyrus, along with damage to the adjacent cortex and subcortical with matter. Most patients with lasting Broca's aphasia, including Broca's original cases, have

much larger left frontoparietal lesions, including most of the territory of the upper division of the left middle cerebral artery. Such patients typically evolve from global to Broca's aphasia over weeks to months. Patients who manifest Broca's aphasia immediately after their strokes, by contrast, have smaller lesions of the inferior frontal region, and their deficits generally resolve quickly. In computed tomography scan analyses have found that lesions restricted to the lower precentral gyrus produced only dysarthria and mild expressive disturbance. Lesions involving the traditional Broca's area (Brodmann's areas 44 and 45) resulted in difficulty initiating speech, and lesions combining Broca's area, the lower precentral gyrus, and subcortical white matter yielded the full syndrome of Broca's aphasia. In studies by the same group, damage to two subcortical white matter sites - the rostral subcallosal fasciculus deep to Broca's area and the periventricular white matter adjacent to the body of the left lateral ventricle was required to cause permanent nonfluency.

Aphemia

A rare variant of Broca's aphasia is aphemia, a nonfluent syndrome in which the patient is initially mute and then able to speak with phoneme substitutions and pauses. All other language functions are intact, including

writing. This rare and usually transitory syndrome results from small lesions either of Broca's area or its subcortical white matter or of the inferior precentral gyrus. Because written expression and auditory comprehension are normal, aphemia is not a true language disorder; aphemia may be equivalent to pure apraxia of speech.

Wernicke's Aphasia

Wernicke’s aphasia may be considered a syndrome opposite of Broca's aphasia, in that expressive speech is fluent but comprehension is impaired. The speech pattern is effortless and sometimes even excessively fluent (logorrhea). A speaker of a foreign language would notice nothing amiss, but a listener who shares the patient's language detects speech empty of meaning, containing verbal paraphasias, neologisms, and jargon productions. Neurolinguists refer to this pattern as paragrammatism. In milder cases, the intended meaning of an utterance may be discerned, but the sentence goes awry with paraphasic substitutions. Naming in Wernicke's aphasia is deficient, often with bizarre, paraphasic substitutions for the correct name. Auditory comprehension is impaired, sometimes even for simple nonsense questions. Auditory perception of phonemes is deficient in Wernicke's aphasia, but deficient semantics is the major cause of the

comprehension disturbance; disturbed access both to semantics and to the internal lexicon is central to the deficit of Wernicke’s aphasia. Repetition is impaired; whispering a phrase in the patient's ear, as in a hearing test, may help cue the patient to attempt repetition. Reading comprehension is usually affected similarly to auditory comprehension, but some patients show greater deficit in one modality. The discovery of spared reading ability in Wernicke's aphasics is important in allowing these patients to communicate. Writing is also impaired, but in a manner quite different from that of Broca's aphasia. The patient usually has no hemiparesis and can grasp the pen and write easily. Written productions are even more abnormal than oral ones, however, in that spelling errors are also evident. Writing samples are especially useful in the detection of mild Wernicke's aphasia.

Associated signs are limited in Wernicke's aphasia; most patients have no elementary motor or sensory deficits, although a partial or complete right homonymous hemianopia may be present.

The psychiatric manifestations of Wernicke's aphasia are quite different from different from those of Broca's aphasia. Depression is less common. Many Wernicke's aphasics seem unaware of or unconcerned about their communicative deficits. With time, come patients become angry or

paranoid about the inability of family members and medical staff to understand them. This behavior, like depression, may hinder rehabilitative efforts.

The lesions of patients with Wernicke's aphasia are usually in the posterior portion of the superior temporal gyrus, sometimes extending into the inferior parietal lobule. Damage to Wernicke's area (Brodmann's area 22) has been reported to correlate most closely with persistent loss of comprehension of single words, although others have found only larger temporoparictal lesions in patients with lasting Wernicke's aphasia.. Extension of the lesion into the inferior parietal region may predict greater involvement of reading comprehension. In terms of vascular anatomy, Wernicke's area lies within the territory of the inferior division of the left middle cerebral artery.

Conduction Aphasia

Conduction aphasia is an uncommon but theoretically important syndrome that can be remembered by its striking deficit of repetition. Most patients have relatively normal spontaneous speech, although some make literal paraphasic errors and hesitate frequently for self - correction. Naming may be impaired, but auditory comprehension is preserved. Repetition may

be disturbed to seemingly ridiculous extremes, such that a patient who can express himself or herself at a sentence level and comprehend conversation may be unable to repeat even single words.. Associated deficits include hemianopia in some patients; right-sided sensory loss may be present, but right sided hemiparesis is usually mild or absent. Some patients have limb apraxia, creating a misimpression that comprehension is impaired. The lesions of conduction aphasia are usually in either the superior temporal or inferior parietal regions. Benson et al. suggested that patients with limb apraxia have parietal lesions, whereas those without apraxia have temporal lesions [15]. Conduction aphasia may represent a stage of recovery in patients with Wernicke's aphasia in whom the damage to the superior temporal gyrus is not complete.

Conduction aphasia has been advanced as a classic disconnection syndrome. Wernicke originally postulated that a lesion disconnecting Wernicke's and Broca's areas would produce this syndrome. Geschwind later pointed to the arcuate fasciculus, a white matter tract traveling from the deep temporal lobe, around the sylvian fissure to the frontal lobe, as the site of disconnection. Anatomic involvement of the arcuate fasciculus is present in most, if not all, cases of conduction aphasia, but there is usually also cortical involvement of the supramarginal gyrus or temporal lobe. The

supramarginal gyrus appears to be involved in auditory immediate memory and in phoneme perception related to word meaning, as well as phoneme generation [39]. Lesions in this area are associated with conduction aphasia and phonemic paraphasic errors.

Global Aphasia

Global aphasia may be thought of as a summation of the deficits of Broca's aphasia and Wernicke's aphasia. Speech in nonfluent or mute, but comprehension is also poor, as are naming, repetition, reading, and writing. Most patients have dense right hemiparesis, hemisensory loss, and often hemianopia, although few patients have little hemiparesis. Milder aphasic syndromes in which all modalities of language are affected are often called mixed aphasias. The lesions of patients with global aphasia are usually large, involving both the inferior frontal and superior temporal regions, and often much of the parietal lobe in between. This lesion represents most of the territory of the left middle cerebral artery. Patients in whom the superior temporal gyrus is spared tend to recover their auditory comprehension and to evolve toward the syndrome of Broca's aphasia. Recovery in global aphasia may be prolonged; global aphasics may recover more during the second month than in the first 6 months after a stroke.

Transcortical Aphasias

The transcortical aphasias are syndromes in which repetition is normal, presumably because the causative lesions do not disrupt the perisylvian language circuit from Wernicke's area through the arcuate fasciculus to Broca's area. Instead, these lesions disrupt connections from other cortical centers into the language circuit (hence the name transcortical). Transcortical motor aphasia is an analogue of Broca's aphasia in which speech is hesitant or telegraphic, comprehension is relatively spared, but repetition is fluent. This syndrome occurs with lesions in the frontal lobe, anterior to Broca's area, and hence within the territory of the anterior cerebral artery. Disruption of the supplementary motor area or disconnection of this area from Broca's area by subcortical frontal white matter lesions may produce the syndrome. The occurrence of transcortical motor aphasia in an arterial territory other than the middle cerebral artery separates this syndrome from the many middle cerebral artery syndromes. Transcortical sensory aphasia, is an analogue of Wernicke's aphasia in which fluent paraphasic speech, paraphasic naming, impaired auditory and reading comprehension, and abnormal writing coexist with normal repetition. This syndrome is relatively uncommon; occurring is strokes of the left temporo-occiptial area and in dementias.

Isolation Aphasia

Mixed transcortical aphasia, or the syndrome of the isolation of the speech area, is a global aphasia in which the patient repeats, often echolalically, but has no comprehension or purposeful speech output. This syndrome is rare, occurring predominantly in large, watershed infarctions of the left hemisphere or both hemispheres that spare the perisylvian cortex or in advanced dementias.

Anomic Aphasia

Anomic aphasia refers to aphasic syndromes in which naming, or access to the internal lexicon, is the principal deficit. Spontaneous speech in normal, except for the pauses and circumlocutions produced by the inability to name. Comprehension, repetition, reading, and writing are intact, except for the same word-finding difficulty in written productions. Anomic aphasia is common but less specific in localization than other aphasic syndromes. Isolated, severe anomia may indicate focal left hemisphere pathology. Alexander and Benson refer to the angular gyrus as the site of lesions producing anomic aphasia, but lesions there usually produce other deficits as well, including alexia and the four elements of Gerstmann's syndrome: agraphia, right-left disorientation, acalulia, and finger agnosia, or inability

to identify fingers. Isolated lesions of the temporal lobe can produce pure anomia, and PET studies of naming in normal subjects have also shown consistent activation of the superior temporal lobe. Inability to produce nouns is characteristic of temporal lobe lesions, whereas inability to produce verbs occurs more with frontal lesions. [38] Even specific classes of nouns may be selectively affected in some cases of anomic aphasia. Anomia is also seen with mass lesions elsewhere in the brain and in diffuse degenerative disorders, such as Alzheimer's disease. Anomic aphasia is also a common stage in the recovery of many aphasic syndromes. Anomic aphasia thus serves as an indicator of left hemisphere or diffuse brain disease, but it has only limited localizing value.

MECHANISMS OF RECOVERY IN POST STROKE APHASIA

Patients with aphasia due to stroke generally show spontaneous improvement over days, weeks, and months. In general greatest recovery occurs during the first 3 months but improvement may continue over a prolonged period, especially in global aphasics [36]. The mechanisms of language recovery have been a subject of several studies. Studies of language activation PET and SPECT scanning techniques are advancing our understanding of neuroanatomy of language recovery. The resolution of acute cellular derangements like oedema, and restoration of blood flow to the ischaemic penumbra explains the recovery in the first few days. The finding of an excessive activation of homologous right sided brain regions, in aphasics compared with normal subject has suggested the role of this non-dominant hemispheric activation in the recovery process. Other studies have suggested that the preserved left hemispheric perilesional area has an important role in recovery of language function [20].

FACTORS INFLUENCING POST STROKE APHASIA

Factors determining the occurrence, severity and outcome of post stroke aphasia remains a subject of controversy. The higher prevalence of stroke in the elderly, and the increasing rate of stroke survival has contributed to the increasing prevalence and incidence of aphasia in the geriatric age group. The effect of age has been attributed to age-related changes in the organization of language areas in some studies while others related to its influence on the vascular pathology and consequently on infarct locations. Some studies have suggested a poorer outcome in older patients.

A much debated question is whether sex differences exist in the functional organization of the brain for language. Language functions are hypothesized to be more likely to be highly lateralized in males and to be represented in both cerebral hemispheres in females, and this has been supported by studies using functional MRI to map brain areas involved in language function [27]. It has been suggested that more bilateral representation of language functions in the female brain may account for this greater improvement in language improvement in female aphasic patients [28].

The study on the Main Characteristics of Patients Hospitalized in Acute Stroke Units by O. Godefroy et al. for the Lille Stroke Program From the Department of Neurovascular Disorders of Lille and Institute d'Orthophonie de Tours, Amiens, France, studied 308 patients [7]. The aim of this study was to evaluate aphasia characteristics at the acute stage in patients admitted to a stroke unit. The mean age of patients was reported as 62 years with a prominence of male sex. Most patients were right handed. Global and nonclassified aphasias accounted for 50% of all aphasic syndromes. Wernicke's, Broca's, and subcortical and transcortical motor aphasia were less frequent, accounting for 40%. Age (P=0.6), sex (P=0.8), and handedness (P=0.4) did not differ across syndromes except in ischemic stroke patients where those with conduction aphasia were younger, and patients with subcortical aphasia were older. The study concluded that that vascular aphasias are frequently severe at the acute stage, that the age effect is due mainly to its influence on infarct location; and that the main determinant of aphasia characteristics is lesion location.

Kertesz and Sheppard examined the sex differences and age distribution in various types of aphasia, in patients with left hemisphere infarct [25]. The higher male to female ratio of aphasia was shown to relate to a similar sex distribution in infarcts rather than to sex differences in

cerebral organization. Broca's aphasics were somewhat younger than the other groups and the slight difference may be related to pathophysiological factors favouring embolic strokes in the anterior territory of the middle cerebral artery. Sex and aphasia types were not significantly different in various age groups.

Heir et al studied the gender differences in aphasia [26]. There were no gender differences in aphasia incidence among the intracerebral hemorrhages. Aphasia was more frequent among women with infarcts (37.0%) than men (28.3%). When stroke site was controlled for, there were no gender differences in aphasia frequency. Wernicke's, global, and anomic aphasias were more common in women than men; Broca's aphasia was somewhat more common in men. Although gender differences were small, the infarct lesions producing aphasia in men were more posteriorly placed and the infarct lesions in women were more anteriorly placed, suggesting possible gender differences in the positioning of the language zone in the brain.

The study on Aphasia in acute stroke: incidence, determinants, and recovery by Pederson et al, Department of Neurology, Bispebjerg Hospital, Copenhagen, Denmark in 1995, studied prospectively and consecutively an

unselected and community-based sample of 881 patients with acute stroke [6]. Assessment of aphasia was done at admission, weekly during the hospital stay, and at a 6-month follow-up using the aphasia score of the Scandinavian Stroke Scale. Thirty-eight percent had aphasia at the time of admission; at discharge 18% had aphasia. Sex was not a determinant of aphasia in stroke, and no sex difference in the anterior-posterior distribution of lesions was found. A valid prognosis of aphasia could be made within 1 to 4 weeks after the stroke depending on the initial severity of aphasia. Initial severity of aphasia was the only clinically relevant predictor of aphasia outcome. Sex, handedness, and side of stroke lesion were not independent outcome predictors, and the influence of age was minimal.

The study on Aphasia after stroke: type, severity and prognosis. The Copenhagen aphasia study, by the same authors (Pedersen PM, Vinter K, Olsen TS. from the Department of Neurology, Bispebjerg Hospital, Copenhagen, Denmark) in 2004 aimed to study the types, severity and evolution of aphasia in unselected, acute stroke patients [21]. 270 acute stroke patients with aphasia (203 with first-ever strokes) were included consecutively and prospectively, and assessed with the Western Aphasia Battery. The assessment was repeated 1 year after stroke. The frequencies of the different types of aphasia in acute first-ever stroke were: global 32%,

Broca's 12%, isolation 2%, transcortical motor 2%, Wernicke's 16%, transcortical sensory 7%, conduction 5% and anomic 25%. These figures were not substantially different from what had been found in previous studies. The type of aphasia always changed to a less severe form during the first year. Nonfluent aphasia could evolve into fluent aphasia (e.g., global to Wernicke's and Broca's to anomic), whereas a fluent aphasia never evolved into a nonfluent aphasia. One year after stroke, the following frequencies were found: global 7%, Broca's 13%, isolation 0%, transcortical motor 1%, Wernicke's 5%, transcortical sensory 0%, conduction 6% and anomic 29%. The outcome for language function was predicted by initial severity of the aphasia and by the initial stroke severity (assessed by the Scandinavian Stroke Scale), but not by age, sex or type of aphasia

Kertesz A, McCabe P studied the Recovery patterns and prognosis in aphasia [14].Ninety-three aphasics were studied with repeated language assessment by a scorable test (the Western Aphasia Battery). Recovery rates were determined by measuring language performance (Aphasia Quotient) at nought to forty-five days post-onset, and three, six and twelve months and yearly after. Recovery rates were higher in post-traumatic than in cerebrovascular cases. Maximum recovery occurred in the first three months. When the stable infarcts were separately studied, the greatest

recovery was seen in "Broca's" aphasics, who frequently evolved into Anomic. Initial severity and outcome correlated significantly. Age and rate of initial recovery showed a trend of negative correlation; younger patients recovered better, but there were frequent exceptions, depending on other factors, such as the initial severity of aphasia. Although some cases recovered exceptionally well, there was no significant difference between the treated and untreated groups, where such a comparison was possible.

DT Wade et al studied 976 patients registered as suffering an acute stroke to determine the natural history of speech disturbance [5]. Of the 545 patients assessed within 7 days of stroke, 24% were aphasic and 28% unassessable. Of those patients aphasic within 7 days, 40% remained so at 6 months; 60% of those aphasic at 3 weeks remained so. There was a high correlation between early and late aphasia scores. Aphasia was associated with more severe disability (degree of limb weakness, loss of function, loss of IQ), and with a less good recovery of social activities.

Pashek and Holland prospectively studied 43 patients with aphasia using the Western Aphasia Battery [23]. They found that 19 out of the 32 patients followed up had a change in aphasia type and that younger patients were more likely to change type.

W Lendrem et al described the spontaneous recovery of language abilities of 52 stroke patients who were aphasic for more than 4 weeks[24]. These patients had been randomly allocated to receive no speech therapy and had been assessed at 6-weekly intervals after a stroke. There was improvement in language abilities over time. Age, sex and aphasia type were not related to the amount of improvement. An aphasic patient's level of language ability at 6 months could be predicted on the basis of the test score on the Porch Index of Communicative Ability at 4 weeks.

McDermott et al. studied the Evolution of acute aphasia using the Western Aphasia Battery [22]. Among the 39 subjects studied initially 13 had Global aphasia, 7 had Wernicke's aphasia, 6 had Brocas aphsia, 5 had anomic aphasia, 5 had transcortical aphasia. At the end of 6 months, 26 (67%) experienced a change in aphasia type. The pattern was Wernicke's 3, anomic 14, global 4, Broca's 9, Conduction 6 and transcortical aphasias 2.Mean AQ including all patients was 15.8.Mean AQ for patients whose aphasia evolved was 19.7, whereas for patients who did not evolve it was 8. Aphasia change scores were significantly associated with age of patient, with older patients showing lesser recovery but not gender. Patients whose aphasia type changed had significantly higher AQ change score than those who did not suggesting that a considerable improvement in language

function was necessary for a evolution of aphasia type. Fluent and non fluent aphasias improved to a similar extent. Baseline AQ was not significantly associated with change in aphasia type, suggesting that initial severity of aphasia was not the only factor determining outcome of language function.

Thus there are some differences of opinion on the influence of age, sex and stroke severity on aphasia and its outcome and this study aims to examine this issue further.

MATERIALS AND METHODS

This study enrolled patients admitted with acute stroke in the Institute of Internal Medicine of Government General Hospital, Chennai during the period from July 2003 to January 2005.

Inclusion criteria

Duration of stroke less than 2 weeks

Single stroke (either ischemic or hemorrhagic)

Confirmation by CT or MRI

Exclusion criteria

Multiple or bilateral stroke

History of prior stroke

Head trauma

Delirium or Dementia

Previous psychiatric illness

Other causes of focal neurological deficits like tumour, infection, inflammation.

Significant hearing or visual impairment

Informed consent was obtained from patients or relatives of patients included in the study. In all patients epidemiological data including age, sex, and presence of risk factors for stroke like hypertension, diabetes, heart disease, smoking, previous TIA were analysed. Patient's handedness as well as educational status were noted.

A general physical examination and neurological examination was done in all patients. Cardiovascular system examination was done to look for source of embolism. The Glasgow coma scale was assessed in all patients. The Glasgow Coma Scale provides a score in the range 3-15. The total score is the sum of the scores in three categories -Eye Opening Response, Verbal Response, and Motor Response [29].

All patients had routine biochemical investigations done. CT Brain was done to identify the pathology--infarct or hemorrhagic stroke, to identify the side of lesion (right vs. left), the site (anterior vs. posterior circulation), and to rule out other causes of focal neurological deficit.

The stroke severity was assessed using the NIH stroke scale. The NIH Stroke Scale is an 11-item clinical evaluation instrument, widely used in clinical trials and practice to assess neurological outcome and degree of recovery. This instrument's reliability [30] and validity [32] are well

documented in scientific literature. The scale includes assessment of the following

1. a. Level of Consciousness
1. b. Ask patient the month and their age
1. c. Ask patient to open and close eyes
2. Best Gaze (only horizontal eye movement)
3. Visual Field Testing
4. Facial Paresis
5. Motor Function - Arm
6. Motor Function –Leg
7. Limb Ataxia
8. Sensory Function
9. Best Language
10. Dysarthria
11. Extinction and Inattention

It gives a 42-point score [32] and classifies severity of stroke as

NIHSS 0 - normal examination

NIHSS 1-7 - mild neurological deficits

NIHSS 8-14 - moderate neurological deficits

NIHSS >=15 - severe neurological deficits

ASSESSMENT OF LANGUAGE FUNCTION

Bedside assessment of language was done for all patients.

Bedside language examination

1. Spontaneous speech
    a. Informal interview
    b. Structured task
    c. Automatic sequences
2. Naming
3. Auditory comprehension
4. Repetition

The first step is to listen to the patient's spontaneous speech .A speech sample is elicited by asking the patient to describe the weather or the reason for coming to the doctor. The following observations are made- fluency, initiation difficulty, articulation, phonation, rate of speech, prosody, and phrase length, presence of circumlocution, word finding pauses, errors such as literal and verbal paraphasias and neologisms.

Fluency was assessed further using the animal naming test- ask the patient to name as many animals as he/she can in 60 seconds. Normal individuals can name 18-22 animals during a 60-second period. Score of less than 13 suggests impairment in verbal fluency [33].

Naming ability is the one of the earliest acquired and most basic of language functions. Anomia was tested with confrontation naming test. A variety of objects or pictures are pointed to and the patient is asked to name them. Several categories of objects are used (colours, body parts, articles of clothing, and parts of objects) since patients may have anomia for a specific category of objects.

Comprehension was tested by asking patients specific questions that can be answered with a “yes” or “no” response or by asking patients to point out various objects in the room. Motor commands are avoided because many patients with aphasia may have apraxia.

Repetition of spoken language is a linguistically and anatomically distinct function. It was tested by presenting material in ascending order of difficulty, beginning with single monosyllabic words and proceeding to complex sentences. Normal people can usually repeat sentences of 19 syllables or 6 words accurately. [34]

Reading and Writing were not assessed in this study since they are directly influenced by educational experience of the patient.

In patients who were found to be aphasic by clinical language examination, language function was assessed in detail based on the Western Aphasia Battery (Kertesz, 1979, 1982)[35]. The Western Aphasia Battery, developed by Kertesz and Poole, as a modified version of the Boston Diagnostic aphasia examination, provides insight into the patient's speech and language functioning and allows grouping of patients into the various aphasia syndromes. The WAB has four subtests-spontaneous speech (fluency and information content), comprehension, repetition and naming. The spontaneous speech subtest carries a maximum score of 20 while other three subtests have a maximum score of 10 each.

The Aphasia Quotient is computed using the subtest scores (the sum of all the subtest scores multiplied by two). Language is classified as normal if AQ of 93.8 or more is achieved[35]. Patient's sub scores on fluency, comprehension, repetition and naming permit classification of language impairment according to the taxonomic table. These discreet cut off scores in the WAB criteria for classification are based on Kertesz's review of 150 patients [35].

Taxonomic Classification of the Western Aphasia Battery

Criteria For Classification

	FLUENCY (0-10)	COMPRE-HENSION (0-10)	REPETITION (0-10)	NAMING (0-10)
GLOBAL	0-4	0-3.9	0-4.9	0-6
BROCA'S	0-4	4-10	0-7.9	0-8
ISOLATION	0-4	0-3.9	5-10	0-6
TRANSCORTICAL MOTOR	0-4	4-10	8-10	0-8
WERNICKE'S	5-10	0-6.9	0-7.9	0-9
TRANSCORTICAL SENSORY	5-10	0-6.9	8-10	0-9
CONDUCTION	5-10	7-10	0-6.9	0-9
ANOMIC	5-10	7-10	7-10	0-9

The questionnaire was translated in Tamil, which is the native language of the subjects included in the study. The battery was first administered during hospital stay (between 7-14 days of stroke onset). Patients were advised follow up every month after discharge. Language function was assessed again after 3 months and for a third time after 6 months of the initial stroke using the same battery.

Data was collected and statistical analysis was done. Chi-square, Student –T and ANOVA tests were used to test the significance of association between the variables. p value of less than 0.05 was taken as statistically significant.

OBSERVATIONS

128 patients with acute stroke were enrolled in this study.

The maximum numbers of patients with stroke were between 60-71 yrs of age. The mean age was 60.22 years.

Age group	No.of patients	Percentage
<40	6	4.7%
41-50	17	13.3%
51-60	41	32.0%
61-70	43	33.6%
>70	21	16.4%

The majority were males (68.8%). Females constituted 31.2% of cases.

Sex	No.of Patients	Percentage
Male	88	68.8%
Female	40	31.2%

28.9% of patients had hypertension while 21% were diabetics. 9.4% had history of heart disease (including ischemic or rheumatic). 31.1% of patients were smokers. Only 3.9% had a history suggestive of a previous TIA.

Risk Factors	No.of Patients	Percentage
Hypertension	37	28.9%
Diabetes Mellitus	27	21.1%
Heart disease	12	9.4%
Smoking	36	28.12%
Transient ischemic attacks	5	3.9%

Only three of the 128 patients were left handed, the remaining being right handed. All three left handed patients had a right hemisphere stroke with normal language function.

Handedness	No.of Patients	Percentage
Right	125	97.7%
Left	3	2.3%

Ischemic strokes constituted the majority -68.8% of cases. 31.3% of patients had a intracranial haemorrhage.

58 patients (45.3%) had right hemisphere lesions whereas

63 patients (49.2%) had left hemisphere lesions.

7 patients had a posterior circulation stroke.

		No.of Patients	**Percentage**
Pathology	Infarct	88	68.8%
	Hemorrhage	40	31.3%
Lesion side	Right	58	45.3%
	Left	63	49.2%
	Posterior	7	5.5%

Most of the patients had a severe neurological deficit as assessed by NIH score.

	NIH Score	**No.of Patients**	**Percentage**
Mild neurological deficit	1-7	5	3.9%
Moderate	8-14	30	23.4%
Severe	>15	93	72.6%

37 patients were excluded from the study because of premature death or discharge. 8 of the patients examined on first visit were later lost to follow up.

Outcome	No.of Patients	Percentage
Alive	83	64.8%
Death / Discharge	37	28.9%
Loss to follow-up	8	6.3%

Among the 91 patients with acute stroke included in this study, 41 patients had aphasia

A majority of patients had global aphasia -25 patients

8 patients had Wernickes aphasia

5 patients had Broca's aphasia

2 patients had Anomic aphasia

1 patient had a Transcortical motor aphasia.

Aphasia type	No.of Patients	Percentage
Global	25	60.9%
Wernicke	8	19.5%
Broca	5	12.1%
Anomic	2	4.8%

Transmotor cortical	1	2.4%

There was a significantly higher prevalence of hypertension and heart disease in stroke patients with aphasia compared to patients with stroke and normal language function. The mean age of aphasic patients (60 yrs) was slightly older than the other patients and there was a male predominance, but both these data were not statistically significant ($p>0.05$).

Characteristic		**N**	**Mean**	**Std. Deviation**	**t test**	**p value**
Age	Normal Language	50	58.02	10.241	0.91	0.36
	Aphasics	41	60.17	12.253		

Characteristic		**Normal language**		**Aphasic patients**		**Chi-sq**	**p value**
Sex	Male	35	70.0%	25	61.0%	0.816	0.36
	Female	15	30.0%	16	39.0%		
Hypertension	Yes	7	14.0%	16	39.0%	7.46	0.006
	No	43	86.0%	25	61.0%		
Diabetes mellitus	Yes	7	14.0%	12	29.3%	3.17	0.07
	No	43	86.0%	29	70.7%		
Heart disease	Yes	3	6.0%	9	22.0%	5.06	0.02
	No	47	94.0%	32	78.0%		
Smoking	Yes	7	14.0%	5	12.2%	0.06	0.8
	No	43	86.0%	36	87.8%		

There was no significant difference in the age and sex distribution in patients with different aphasia syndromes though patients with Broca's aphasia were slightly younger(mean age 52 yrs) than patients with Wernicke's and Global aphasia (mean age 61 yrs).

Aphasia type	**No.of Patients**	**Mean Age**	**Std. Deviation**	**ANOVA**
Global	25	61.40	13.823	F=1.18 p=0.32
Wernicke	8	60.88	11.064	
Broca	5	52.00	5.477	
Total	38	60.05	12.668	

Sex	**Aphasia_type**			**Total**	**Chi square test**
	Global	**Wernicke**	**Broca**		
Male	6	5	4	23	$\chi^2 = 1.2$ p = 0.6
Female	11	3	1	15	
Total	25	8	5	38	

In the 33 patients who were followed up, 11(33%) patients had a change in the type of aphasia at the end of 6 months.

Among the 25 patients with global aphasia – 5 evolved into Wernicke's aphasia and 3 patients evolved into a Broca's aphasia.

Among the patients with Broca's aphasia, one evolved into an anomic aphasia.

There was no change in type in patients with Wernicke's aphasia

One patient with anomic aphasia had normal language function at the end of 6 months.

The lone patient with a transmotor cortical aphasia evolved into an anomic aphasia.

At the end of the period of follow up of the 33 patients, 10 patients had global aphasia (30%), 13 had Wernickes aphasia (39%), 7 had Broca's aphasia (20%) while 3 patients had anomic aphasia (1%).

Aphasia type	**No.of Patients**	**Percentage**
Global	10	30.3%
Wernicke	13	39.4%
Broca	7	21.2%
Anomic	3	9.0%
Trans motor	0	0

Comparing the patients whose aphasia evolved to those whose remained the same at the end of 6 months there was no significant difference in age. More females had a change in aphasia type compared to males.

Aphasia	**No.of Patients**	**Mean age**	**Std. Deviation**	**t - test**	**p - value**
Aphasia type not changed	22	61.81	12.032	0.52	0.60
Aphasia type changed	11	59.36	12.987		

Sex	**No.of patients aphasia not evolved**	**Percentage**	**No.of patients aphasia evolved**	**Percentage**	**Chi Square**
Male	16	71.4%	4	36.4%	$\chi^2 = 3.67$ **p = 0.04**
Female	6	28.6%	7	63.6%	

The change in AQ (final AQ-initial AQ) was significantly higher in patients whose aphasia type changed. The initial AQ was also higher in patients who evolved though not statistically significant.

	No.of Patients	**Mean**	**Std. Deviation**	**t - test**	**p - value**
AQ Change					
Aphasia not evolved	22	9.1333	3.91016	4.81	**0.001**
Aphasia type evolved	11	17.3455	5.69146		
Initial AQ					
Aphasia not Evolved	22	26.17	22.240	0.53	0.59
Aphasia Evolved	11	30.55	21.311		

The initial severity of stoke as measured by NIH Scale was not significantly different in patients whose aphasia type changed compared to those whose aphasia type remained the same.

	No.of Patients	**Mean**	**Std. Deviation**	**t - test**	**p - value**
NIH score					
Aphasia not Evolved	22	18.6	6.08	1.15	0.26

Aphasia Evolved	11	16.54	4.01		

Correlating the change in AQ to the initial severity of stroke as measured by the NIH scale, there was a significant negative correlation (Pearsons Correlation coefficient r = - 0.86, p = 0.001)

Comparing the AQ change in various types of aphasia the maximum gain in AQ occurred in Broca's aphasics and minimum in Wernicke's aphasics.

Type of aphasia	**Number of patients**	**Mean AQ change**	**Std deviation**	**F test**
Global	25	12	4.7	F=2.5 P=0.07
Wernicke's	8	8	4.2	
Broca's	5	18	7.7	
Anomic	2	11.3	10.32	
Transmotor	1			

The average AQ change in the first 3 months (AQ at 3rd month –initial AQ) was 8.62, which was higher than the average AQ change over the next three months (AQ at 6th month –AQ at 3rd month) which was 3.43 .Of the 11

patients whose aphasia evolved during the 6 month study period, 9 patients had a change in type of aphasia by the end of 3 months. Only 2 patients had a change in aphasia type between the third and sixth month of follow up.

DISCUSSION

The mean age of patients in this study was 60 years with maximum patients in their 6th decade. The majority (68.8%) were males. Indian studies like Paithankar et al have reported the mean age as 71.3 and the maximum number of patients in the seventh decade with a male predominance [37]. This data is consistent with other studies, which have reported a higher prevalence of stroke in elderly age group and in males. Most patients enrolled in this study were right handed and this is similar to data from other studies [22].

After the first language evaluation (within 2 weeks of stroke) of patients enrolled in this study, 41 had aphasia (32%). Aphasia has been observed with a frequency ranging from 21% to 38% at the acute stage of stoke [3,6]. A majority of the patients had global aphasia -25 patients (60%). This data is consistent with that of Godefroy et al who reported Global aphasia in 50% of their aphasic patients [7]. Other studies have reported lower incidence [6]. Since this study was done in the inpatients of a tertiary

care hospital, this data does not reflect the actual prevalence of stroke related aphasia in the community.

Broca's aphasia constituted 12% of the cases in this study and Wernicke's 19.5% of cases at presentation. This is consistent with other studies .The Copenhagen aphasia study also reported Broca's in 12% and Wernickes in 16% of their cases and McDermott et al reported Broca's in 15% and Wernicke's in 18% of their cases of aphasia[21,22].In this study, only one patient had Transcortical motor aphasia, and 2 patients had Anomic aphasia. This data is lower than in the studies quoted above. This may be explained by a referral bias and admission policy of the hospital which favours inpatient care for non ambulant stoke patients with severe motor and cognitive deficits.

Comparing the patients with normal language function to those with aphasia, the mean age of aphasic patients (60 yrs) was slightly older than the other patients and there was a male predominance, but both these data were not statistically significant ($p>0.05$). This correlates with data published by Kertesz et al who proposed that the higher male to female ratio of aphasia relates to a similar sex distribution in infarcts rather than to sex differences in cerebral organization [25] .

Among patients with different types of aphasia in this study, there was no significant difference in age ($p>0.05$) though patients with Broca's aphasia were slightly younger (mean age 52 yrs) than patients with Wernicke's and Global aphasia (mean age 60 yrs). Kertesz et al in their study reported that Broca's aphasics were somewhat younger than the other groups and that slight difference in age may be related to pathophysiological factors favouring embolic strokes in the anterior territory of the middle cerebral artery. Godefroy et al reported that age did not differ across the various aphasic syndromes, except that patients with conduction aphasia were younger, and patients with subcortical aphasia were older than patients with other types of aphasia.

There was no significant difference in the sex distribution across various aphasic syndromes in this study. Kertesz et al, Godefroy et al also reported that sex did not significantly differ across aphasic syndromes in acute stroke.

All patients followed up had some improvement in their aphasia quotient. In this study 11of the 33 patients (33%) had a change in type of aphasia. Among the 25 patients with global aphasia – 5 evolved into Wernicke's aphasia while 3 patients evolved into a Broca's aphasia. Among

the patients with Broca’s aphasia, one evolved into an anomic aphasia. There was no change in type in patients with Wernicke’s aphasia. One patient with anomic aphasia had normal language function at the end of 6 months. The lone patient with a transmotor cortical aphasia evolved into a anomic aphasia. This is similar to data from the the Copenhagen aphasia study which concluded that the type of aphasia always changed to a less severe form during the first year. Nonfluent aphasia could evolve into fluent aphasia (e.g., global to Wernicke's and Broca's to anomic), whereas a fluent aphasia never evolved into a nonfluent aphasia [21].

Comparing the patients whose aphasia type changed to those whose aphasia type remained the same at the end of 6 months of study period there was no significant difference in age suggesting that age was not a poor prognostic factor for aphasia recovery. More females had a change in aphasia type compared to males, suggesting that outcome of language function is better in females. According to the Copenhagen aphasia study outcome for language function was not influenced by age, sex or type of aphasia. However Kertesz et al reported that age and rate of initial recovery showed a trend of negative correlation; younger patients recovered better, but there were frequent exceptions, depending on other factors, such as the initial severity of aphasia. Pashek et al also reported that 59% of their

aphasic patients had change in type of aphasia and older patients had a poorer language outcome. Pizzamiglio et al. studied the sex differences in aphasia recovery and reported that although no initial sex difference was found in severity of language disorders, females within the global aphasic group showed significantly greater improvement tests of language [28]. They suggested that the better recovery in females was due to a bilateral representation of language function in the female brain compared to the male brain.

The change in AQ (final AQ-initial AQ) was significantly higher in patients whose aphasia type changed indicating that evolution of aphasia type requires considerable recovery in language function .The initial AQ was also higher in patients whose aphasia evolved though not statistically significant. McDermott et al reported a significant change in aphasia scores in patients whose aphasia had evolved, and a slightly higher initial AQ in patients whose aphasia type changed .They concluded that initial severity (as reflected by initial AQ) was only one of the factors predicting aphasia recovery.

In this study, the increase in AQ in the a first three months following the stroke was higher than in the following three months, indicating that

maximum recovery of language function occurred in the first 3 months. This is consistent with finding reported by Kertesz et al and McDermott et al [14,22]. This improvement may be attributed to the resolution of acute ischemic changes like perilesional edema and restoration of blood flow to the ischemic penumbra allowing these regions of the brain to function effectively.

Correlating the change in AQ to the initial severity of stroke as measured by the NIH scale, there was a significant negative correlation ($p=0.01$). Initial severity of stroke therefore was a good predictor of the extent of recovery of language function. The Copenhagen Aphasia study used the Scandinavian stroke scale, and also found that initial stroke severity was an important factor in determining the language outcome of the patient.

CONCLUSIONS

- Global aphasia is the most common aphasic syndrome in acute stroke patients, followed by Wernicke's aphasia, Broca's aphasia, Anomic aphasia, and Transmotor cortical aphasia. Age and Sex did not differ significantly across the various types of aphasia.

- Recovery in patients with aphasia due to stroke is a dynamic process with evolution of aphasia from one type to another. There was some improvement in language function as reflected by the Aphasia Quotient in all patients followed up. The type of aphasia changed to a less severe form in 33% of patients. Nonfluent aphasia may evolve into fluent aphasia. Global aphasia evolved into either Wernicke or Broca's aphasia. Broca's and Transmotorcortical aphasia evolved into an Anomic aphasia. Anomic aphasia evolved to normal language function. There was no change in aphasia type in patients with Wernicke's aphasia.

- Age did not affect the recovery from aphasia. Recovery was better in women whose aphasia evolved more frequently to a less severe form.

- The extent of recovery was inversely related to the initial severity of stoke as measured by the NIH stroke scale.

- The maximum recovery in language function occurred in the first three months following the stroke.

PROFORMA

Name :

Age :

Sex :

Occupation :

Hospital inpatient no:

Patient's address :

Presenting complaints:

Duration

Inability to usearm arm and leg on one side of the body :

Sensory disturbance/numbness on one side of the body :

Blindness/field defects :

Facial asymmetry :

Difficulty in swallowing/nasal regurgitation/slurred speech /vertigo :

Communication with bystanders :

Bladder/bowel disturbances :

H/o headache vomiting

seizures loss of consiousness

fever

Risk factors:

Hypertension Smoking

Diabetes Alcohol consumption

Heart disease Substance abuse

Symptoms of previous TIA/CVA:

Sudden monocular blindness:

Weakness/clumsinessof arm /leg:

Numbness /unilateral sensory symptoms:

Ataxia : diplopia : vertigo:

Dysphasia: Dysarthria

Visual field defects:

Past history:

Tuberculosis

Arthritis/skin changes/oral ulcers

HIV/Syphilis

Migraine

Peripheral vascular disease

Trauma

Ear discharge

Sinus disease

Seizures

Treatment history:

EXAMINATION

General examination

Vital signs: Pulse: Carotids:

Blood pressure:

Respiratory rate:

Temperature :

Central nervous system examination:

Glassgow coma scale:

Handedness :

Level of education :

Cooperation :

Higher mental function:

Cranial nerves:

Spinomotor system:

State of nutrition of muscles:

	Power		Tone	
	Right	Left	Right	Left
Upper limb				
Lower limb				

Co-ordination:

Involuntary movements:

Reflexes:	Right	Left
Corneal		
Abdominal		
Plantar		
Deep tendon reflexes		
Sensory system		

ANS:

Gait :

Skull and spine:

OTHER SYSTEM EXAMINATION FINDINGS:

DIAGNOSIS: ANTR CIRC POSTR CIRC

PARTIAL COMPLETE

INVESTIGATIONS:

Blood sugar: Sr. sodium: Bl urea :

Sr creatinine:

ECG:

CT Brain :

Lesion-

Location-

NIH STROKE SCALE

1.a. Level of Consciousness:	0 Alert
	1 Not alert, but arousable with minimal stimulation
	2 Not alert, requires repeated stimulation to attend
	3 Coma
1.b. Ask patient the month and their age:	0 Answers both correctly
	1 Answers one correctly
	2 Both incorrect
1.c. Ask patient to open and close eyes and	0 Obeys both correctly
	1 Obeys one correctly
	2 Both incorrect
2. Best gaze (only horizontal eye movement):	0 Normal
	1 Partial gaze palsy
	2 Forced deviation
3. Visual Field testing:	0 No visual field loss
	1 Partial hemianopia
	2 Complete hemianopia
	3 Bilateral hemianopia (blind including cortical blindness)
4. Facial Paresis (Ask patient to show	0 Normal symmetrical movement
teeth or raise eyebrows and close eyes	1 Minor paralysis (flattened nasolabial fold, asymmetry on smiling)
tightly):	2 Partial paralysis (total or near total paralysis of lower face)
	3 Complete paralysis of one or both sides (absence of facial movement in the upper and lower face)
5. Motor Function - Arm (right and left):	0 Normal (extends arems 90 (or 45) degrees for 10 seconds without drift)
	1 Drift
Right arm ____	2 Some effort against gravity
Left arm ____	3 No effort against gravity

	4 No movement
	9 Untestable (Joint fused or limb amputated)
6. Motor Function - Leg (right and left):	0 Normal (hold leg 30 degrees position for 5 seconds)
	1 Drift
Right leg ____	2 Some effort against gravity
Left leg ____	3 No effort against gravity
	4 No movement
	9 Untestable (Joint fused or limb amputated)
7. Limb Ataxia:	0 No ataxia
	1 Present in one limb
	2 Present in two limbs
8. Sensory (Use pinprick to test arems, legs,	0 Normal
trunk and face -- compare side to side):	1 Mild to moderate decrease in sensation
	2 Severe to total sensory loss
9. Best Language (describe picture, name items,	0 No aphasia
read sentences)	1 Mild to moderate aphasia
	2 Severe aphasia
	3 Mute
10. Dysarthria (read several words):	0 Normal articulation
	1 Mild to moderate slurring of words
	2 Near unintelligible or unable to speak
	9 Intubated or other physical barrier
11. Extinction and inattention:	0 Normal
	1 Inattention or extinction to bilateral simultaneous stimulation in one of the sensory modalities
	2 Severe hemi-inattention or hemi-inattention to more than one modality

NIHSS SCORE -

WESTERN APHASIA BATTERY (modified)

I. SPONTANEOUS SPEECH

1. How are you today?
2. Have you come here before?
3. What is your name?
4. What is your address?
5. What is your job?
6. What is the complaint for which you have come to the hospital?
7. Look at the picture and tell me what you think.

Information Content : 10 Fluency, Grammatical Competence and Paraphasia - 10

Max. Score : 20

Patient Score :

II. AUDITORY VERBAL COMPREHENSION

1. **Yes or No questions** (20 Questions)

Max. Score : 60

Patient Score :

2. **Auditory word recognition:**

Real Objects	Drawn Objects	Forms	Letters	Numbers
Pen	House	Square	P	10
Coin	Bottle	Plus symbol	M	25
Wristwatch	Flower	Circle	A	1438
Purse	Man	Star	O	7
Torch light	Clock	Triangle	K	300
Key	Cup	Arrow mark	U	5

Colours	Furnitures	Body parts	Fingers	Right-Left parts
Red	Window	Ear	Thumb	Right shoulder
Green	Chair	Nose	Ring finger	Left eye
Blue	Table	Eye	Little finger	Right wrist
Black	Door	Jaw	Ring finger	Left ankle
Yellow	Roof	Neck	Index finger	Right ear
Violet	Stool	Chest	Big toe	Left elbow

Max. Score : 60

Patient Score :

3. **Sequential Commands**

1. Lift your hand (2)
2. Close your eyes (2)
3. Show me the chair (2)
4. Show me the door after showing me the chair(4)
5. Show me the pen and the book (4)
6. With the pen point out the book (8)
7. With the pen point out the scale (8)
8. With the scale point out the book (8)
9. With the book point out the pen (8)
10. Give me the book with the pen on it (8)
11. Keep the scale near the pen and book on top of it (20)

Max. Score : 80

Patient Score :

III. REPETITION

		Max. Score	Patient's Score
1.	Bus	2	
2.	Window	2	
3.	Eyes	2	
4.	Nose	2	
5.	Banana	4	
6.	Watch	4	
7	Forty five	4	
8.	Ninety five percent	6	
9.	Two hours and fifty five minutes	10	
10.	The bell is ringing	8	
11.	A ball is rolling near the sea shore	10	
12.	All that glitters is not gold	6	
13.	I went to the zoo to see a tiger	10	
14.	I went to a shop and bought chocolates	10	
15.	Mahabalipuram is situated at a distance of 30km from Parry's corner in Chennai	20	
		---- 100 ----	

Max. Score : 100 Patient Score :

IV. NAMING

a) **Object Naming**

1.	Wrist watch	2.	Pen	3.	Book
4.	Coin	5.	Key	6.	Scale
7.	Torchlight	8.	Inch tape	9.	Purse
10.	Soap box	11.	Match box	12.	Tooth brush
13.	Lock	14.	Comb	15.	Spoon
16.	Ink bootle	17.	Cassette	18.	Tablets
19.	Powder box	20.	Bulb		

Max. Score : 60

Patient Score :

b) **Word Fluency**: In one minute tell me as many animal names as you can

Max. Score : 20

Patient Score :

c) **Sentence Completion** :

1. Milk is …… …. in colour
2. There is …… in the pond.
3. We …….. with a pen
4. We celebrate Pongal in the month of …….
5. Make hay ……..

Max. Score : 10

Patient Score :

d) **Responsive Speech :**

1. What do you write with?
2. What is the colour of the elephant?
3. How many days are there in a week?
4. Who drives the bus?
5. Where can you buy stamps?

Max. Score : 10

Patient Score :

WAB SCORE

	LANGUAGE PARAMETERS	**Maximum score**	**Patient's subscores**	**Total for AQ**
I	SPONTANEOUS SPEECH: Information content Fluency	 10 10		
	Total	20		
II	COMPREHENSION Yes-No questions Auditory Word Recognition Sequential Commands	 60 60 80		
	Total	200		
	(divided by 20 for AQ)	10		
III	REPETITION	100		
	Total	100		
	(divided by 10 for AQ)	10		
IV	NAMING Object Naming Word Fluency Sentence Completion Responsive Speech			
	Total	100		
	(divided by 10 for AQ)	10		

APHASIA QUOTIENT:

$$AQ = \left\{ \begin{array}{l} \text{Spontaneous speech score (max 20)} \\ + \dfrac{\text{Comprehension score}}{20} \text{ (max 10)} + \\ \dfrac{\text{Repetition score}}{10} \text{ (max 10)} + \dfrac{\text{Naming score}}{10} \text{ (max 10)} \end{array} \right\} \times 2$$

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<40	6
41-50	17
51-60	41
61-70	43
>70	21

Male	88
Female	40

Hypertensi	37
Diabetes m	27
Heart disea	12
Smoking	36
Transient is	5

Infarct	88
Hemorrhag	40

Right hemi	58
Left hemisp	63
Posterior c	7

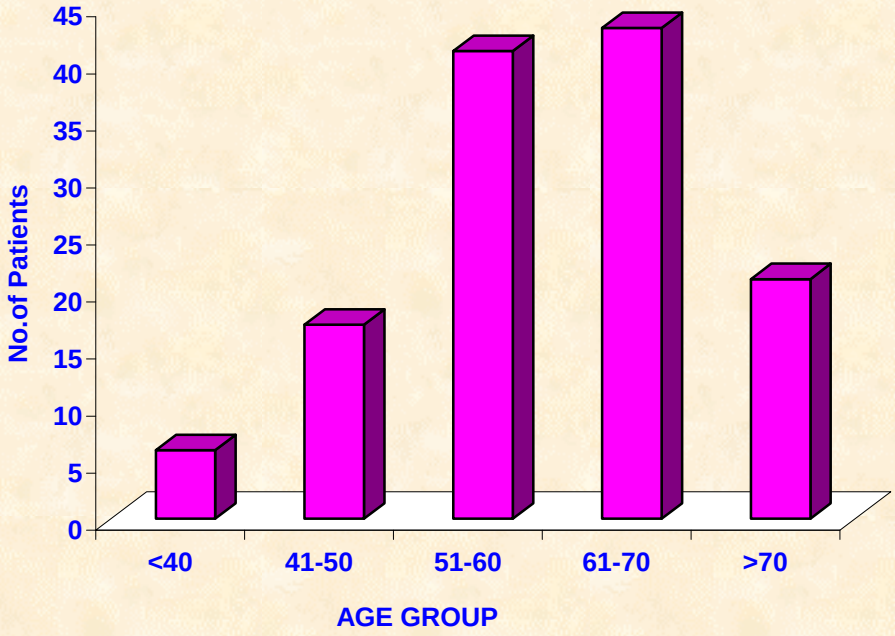
1-7	5
8-14	30
>15	93

Global aph	25
Wernicke's	8
Broca's apl	5

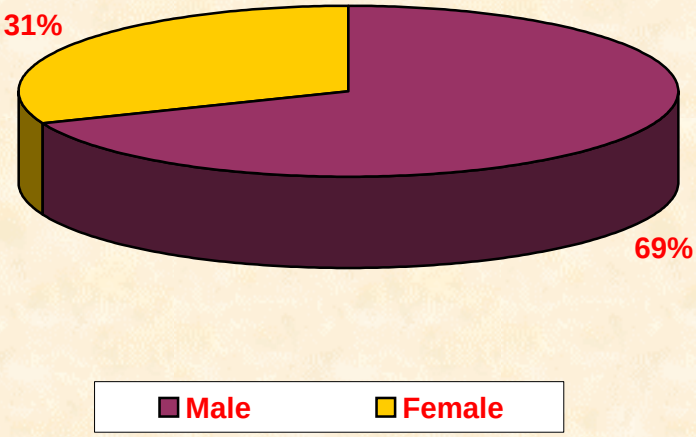
Anomic apl	2
Transmoto	1

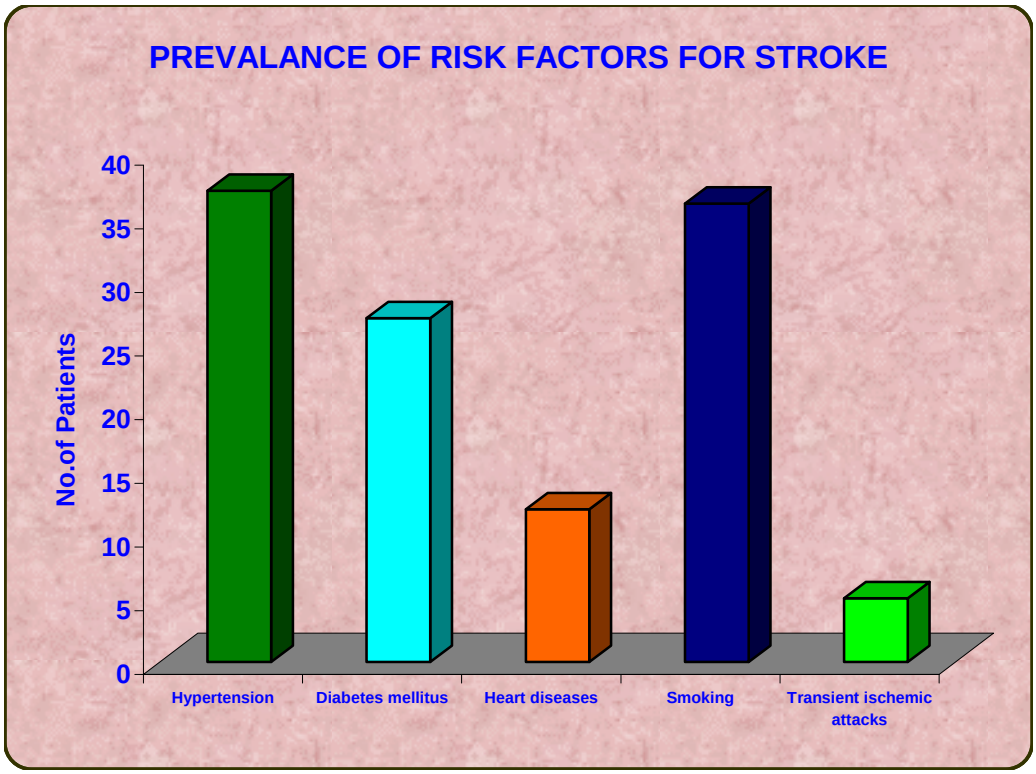
Global aph	10
Wernicke's	13
Broca's apl	7
Anomic apl	3
Transmoto	0

AGE DISTRIBUTION OF STROKE PATIENTS



SEX DISTRIBUTION OF STROKE PATIENTS




PREVALANCE OF RISK FACTORS FOR STROKE
No.of Patients
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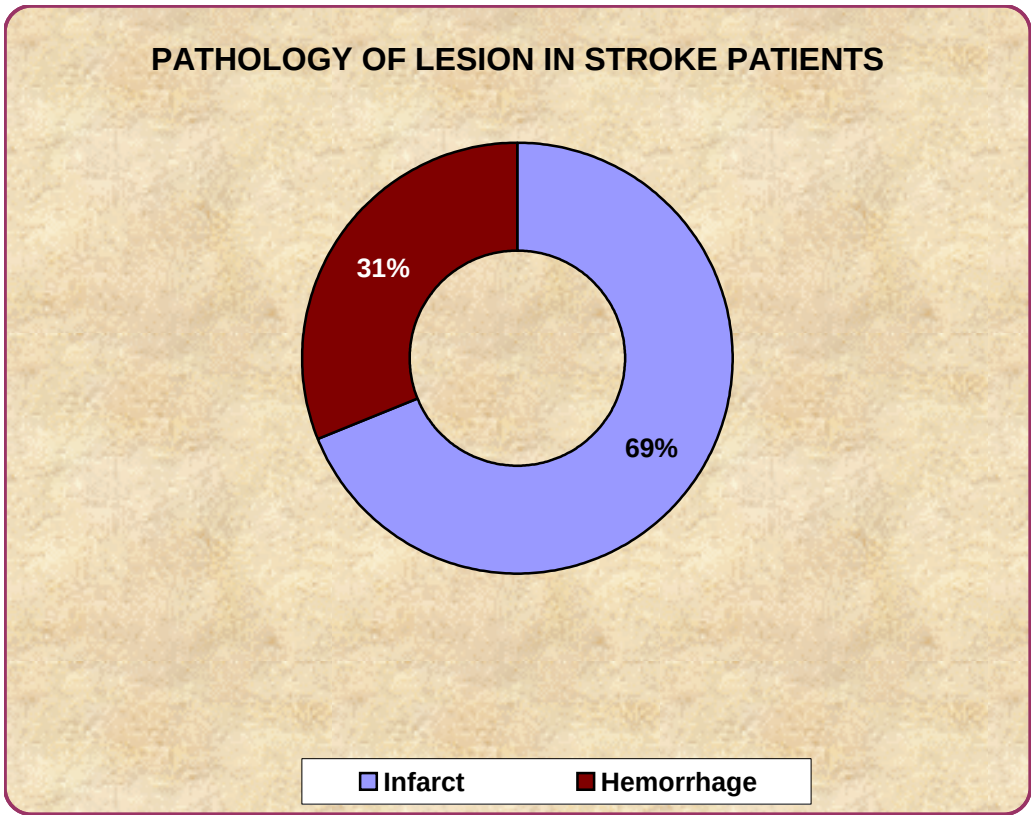
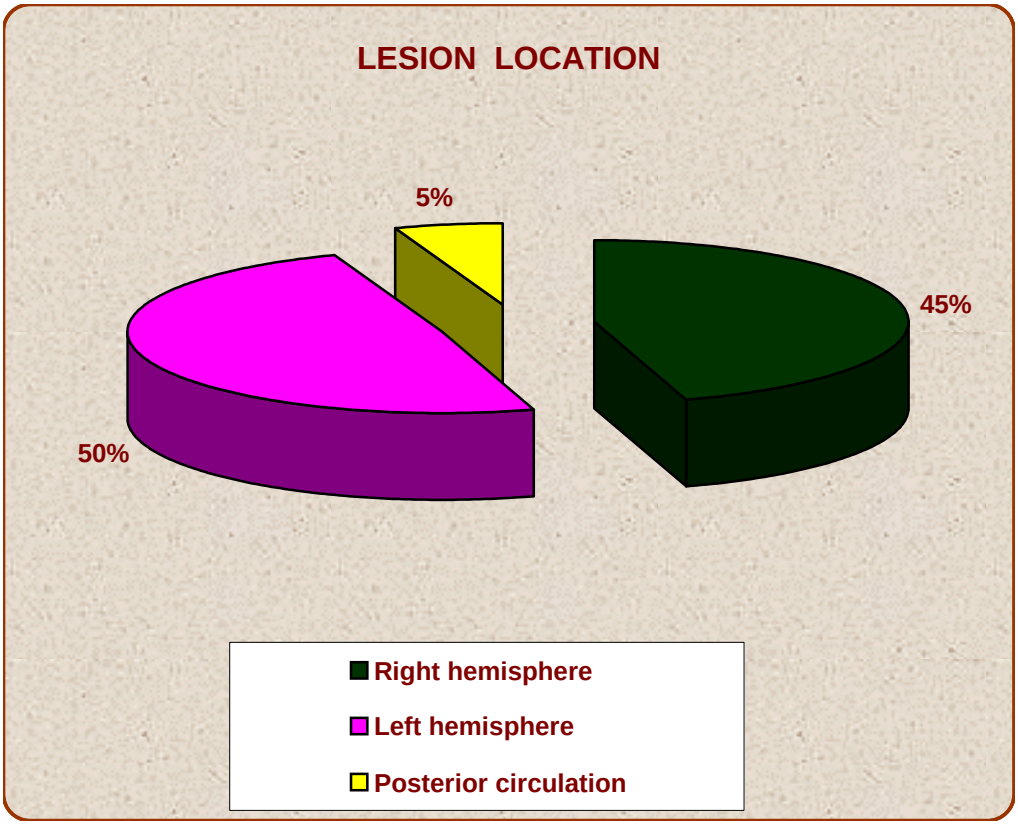
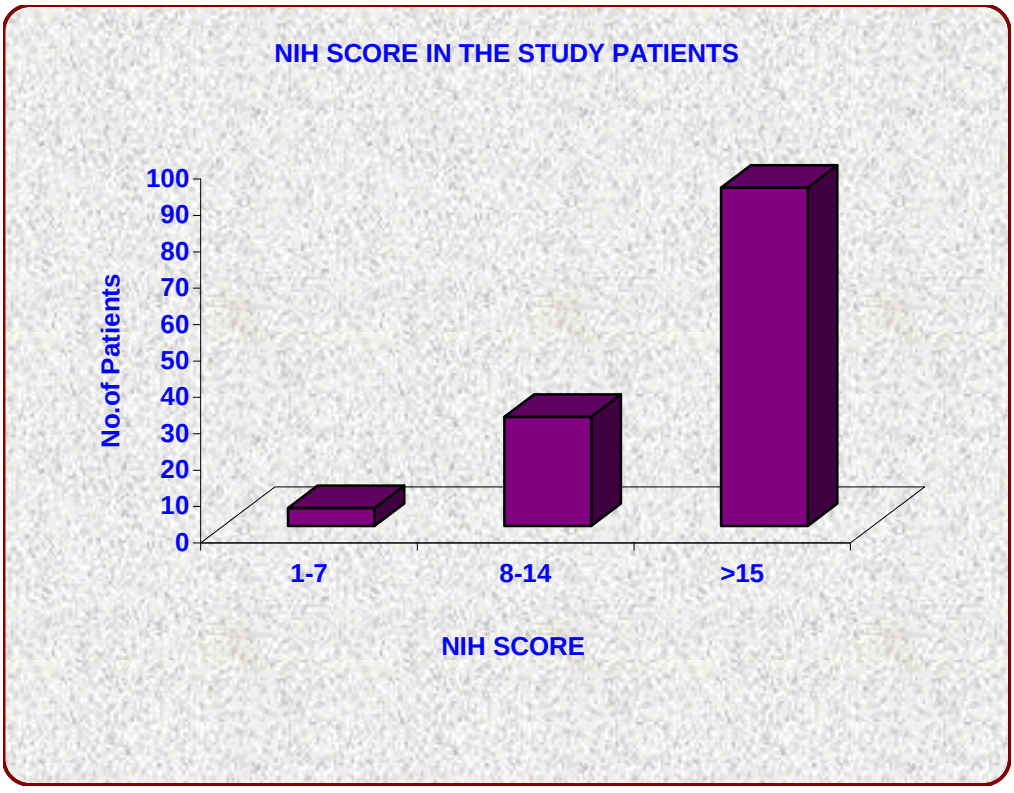
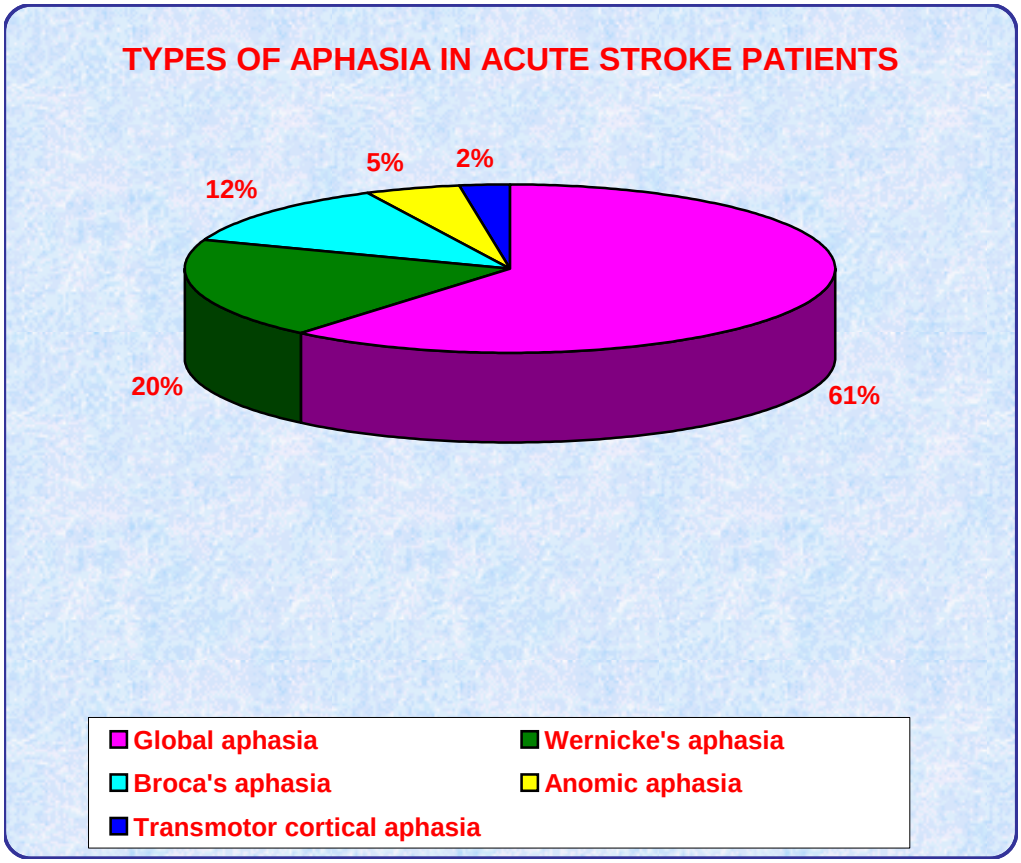
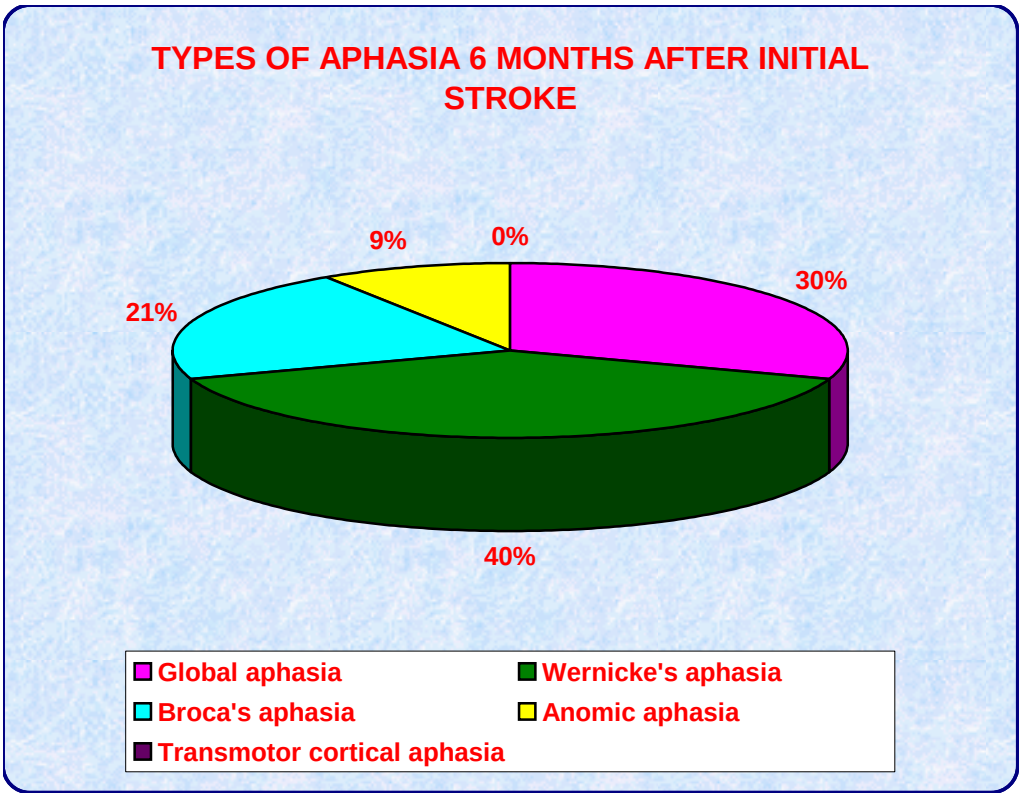
35
30
25
20
15
10
5
0
Hypertension
Diabetes mellitus
Heart diseases
Smoking
Transient ischemic attacks


PATHOLOGY OF LESION IN STROKE PATIENTS
31%
69%
Infarct
Hemorrhage


LESION LOCATION
5%
45%
50%
Right hemisphere
Left hemisphere
Posterior circulation


NIH SCORE IN THE STUDY PATIENTS
No.of Patients
100
90
80
70
60
50
40
30
20
10
0
1-7
8-14
>15
NIH SCORE


TYPES OF APHASIA IN ACUTE STROKE PATIENTS
5%
2%
12%
20%
61%
Global aphasia
Wernicke's aphasia
Broca's aphasia
Anomic aphasia
Transmotor cortical aphasia


TYPES OF APHASIA 6 MONTHS AFTER INITIAL STROKE
9%
0%
30%
21%
40%
Global aphasia
Wernicke's aphasia
Broca's aphasia
Anomic aphasia
Transmotor cortical aphasia

serial no	age	sex	HTN	DM	Heart Dis.	smoking	TIA	BP
1	28	m	n	n	n	y	n	n
2	66	m	y	n	n	n	n	II
3	70	f	n	n	n	n	y	n
4	68	f	n	y	n	n	n	n
5	45	f	n	n	y	n	n	n
6	42	m	y	n	y	n	n	II
7	67	m	n	n	n	n	n	I
8	59	m	y	y	n	n	n	I
9	58	f	n	y	n	n	n	n
10	56	m	y	n	n	n	n	I
11	55	f	n	y	n	n	n	n
12	60	m	n	y	n	y	n	n
13	57	m	n	n	n		n	n
14	61	f	n	n	n	n	n	II
15	62	m	n	n	n	n	n	I
16	66	f	n	y	n	n	n	n
17	45	m	n	y	n	n	n	n
18	59	m	n	y	n	n	n	n
19	68	f	n	n	n	n	n	n
20	44	m	n	n	n	n	n	II
21	56	m	y	n	n	y	n	I
22	42	m	y	y	n	n	y	II
23	71	m	n	y	n	n	y	n
24	66	m	n	n	n	n	n	n
25	67	m	n	n	n	n	n	n
26	78	f	n	n	n	n	n	n
27	61	f	n	n	n	n	n	n
28	54	m	n	n	n	y	n	I
29	68	m	y	n	n	n	n	I
30	51	f	y	n	n	n	n	I
31	78	f	y	n	n	n	n	II
32	67	f	n	n	n	n	n	I
33	43	m	y	y	y	n	n	I
34	66	m	n	n	n	y	n	I
35	44	m	n	n	n	n	n	n
36	58	f	n	n	n	n	n	n
37	76	m	n	n	n	n	n	n
38	55	f	y	n	n	y	y	II
39	56	m	n	n	n	n	n	n
40	71	m	n	n	n	n	n	n
41	34	m	n	n	y	y	n	n
42	72	f	n	n	n	n	n	n
43	57	m	n	n	n	n	n	n
44	78	f	n	n	n	n	n	II
45	60	f	y	n	n	n	n	I
46	39	m	y	n	n	n	n	II
47	55	m	n	n	n	n	n	n
48	62	f	n	n	n	n	n	n
49	54	f	y	n	n	n	n	I

50	67	f	n	n	n	n	n	n
51	79	m	n	n	n	n	n	I
52	59	m	n	n	n	n	n	n
53	62	m	n	n	n	n	n	n
54	66	m	n	n	n	n	n	n
55	61	f	y	n	n	n	n	II
56	62	m	n	n	n	n	n	n
57	59	m	n	n	n	n	n	I
58	61	m	n	n	n	y	y	n
59	47	m	n	n	n	n	n	n
60	53	m	n	n	n	n	n	n
61	48	m	y	y	y	n	n	I
62	76	f	y	n	n	n	n	I
63	73	m	n	y	n	n	n	n
64	67	f	n	y	n	n	n	n
65	46	m	n	n	n	n	n	n
66	59	m	n	n	n	y	n	I
67	57	m	n	y	n	y	n	n
68	81	f	n	n	n	n	n	n
69	57	m	n	n	n	n	n	n
70	65	m	n	n	n	n	n	n
71	67	f	n	n	n	n	n	II
72	48	m	n	n	n	n	n	n
73	67	f	n	n	n	n	n	n
74	55	m	n	n	n	n	n	n
75	80	m	n	n	n	n	n	n
76	32	m	n	n	y	n	n	n
77	67	f	n	y	n	n	n	n
78	58	m	n	n	n	n	n	n
79	68	m	n	n	n	n	n	n
80	55	m	y	y	n	n	n	II
81	53	m	n	n	n	n	n	n
82	68	f	n	n	n	n	n	n
83	60	m	n	n	n	n	n	n
84	59	f	n	n	n	n	n	II
85	68	f	n	y	n	n	n	n
86	76	m	n	n	n	n	n	n
87	52	m	y	n	n	n	n	II
88	69	m	y	y	n	n	n	I
89	75	n	y	n	n	y	n	n
90	70	f	n	n	n	n	n	n
91	44	m	n	n	n	n	n	II
92	83	m	n	n	n	n	n	n
93	65	m	n	y	n	n	n	n
94	57	f	n	n	n	n	n	n
95	63	m	n	n	n	n	n	II
96	76	f	y	n	n	n	n	n
97	53	m	y	n	n	y	n	II
98	80	m	n	n	n	n	n	n
99	70	m	y	n	n	n	n	n
100	56	m	y	y	n	n	n	II

101	54	m	y	n	n	n	n	I
102	67	m	n	n	n	n	n	n
103	63	f	n	n	n	n	n	n
104	32	f	n	n	y	n	n	n
105	44	m	y	n	n	y	n	II
106	66	f	n	n	n	n	n	n
107	61	m	n	n	n	n	n	n
108	49	f	y	y	n	n	n	II
109	46	m	y	n	y	n	n	I
110	57	m	n	n	n	n	n	I
111	73	m	y	n	n	n	n	II
112	57	m	y	n	n	n	n	I
113	63	m	n	n	n	n	n	n
114	39	m	n	y	n	n	n	n
115	67	m	n	y	n	n	n	n
116	55	m	y	n	y	n	n	n
117	82	m	n	n	n	n	n	n
118	55	m	n	n	y	n	n	n
119	53	m	n	y	y	n	n	n
120	67	m	y	n	n	n	n	I
121	52	m	y	n	n	n	n	II
122	49	n	n	n	n	n	n	II
123	75	m	n	n	n	y	n	n
124	48	f	y	y	n	n	n	I
125	56	m	y	n	n	n	n	II
126	62	m	n	n	y	n	n	n
127	70	f	n	n	n	n	n	n
128	72	m	y	y	n	n	n	n

gcs	NIHscore	handed	inf/hage	lesionside	teritory	outcome	WAB score first exami fluency	l spon sp
10	20	rt	i	lt	ant	a	0	0
5	32	rt	h	lt	ant	d		
12	22	rt	i	lt	ant	a	0	0
7	34	rt	i	rt	ant	d		
12	14	rt	i	lt	ant	a	10	20
10	24	rt	i	lt	ant	l	1	1
14	17	rt	i	lt	ant	a	10	20
5	36	rt	i	rt	ant	d		
9	20	lt	i	rt	ant	a	10	20
9	22	rt	h	lt	ant	l	0	0
12	27	rt	i	rt	ant	a	10	9
13	13	rt	i	lt	ant	a	2	10
4	34	rt	i	rt	ant	d		
8	16	rt	i	rt	ant	a	9	18
9	27	rt	h	rt	ant	a	9	18
10	25	rt	i	lt	ant	a	2	6
12	22	rt	i	lt	ant	l	0	0
13	20	rt	i	rt	ant	a	10	20
13	22	rt	i	lt	ant	a	10	20
10	23	rt	h	rt	ant	a	10	20
14	13	rt	i	lt	ant	a	2	6
5	34	rt	h	rt	ant	d		
10	16	rt	h	rt	ant	a	10	20
12	23	rt	i	lt	ant	a	10	20
4	36	rt	h	post	post	d		
9	26	rt	i	lt	ant	l	0	0
13	26	rt	i	rt	ant	a	9	18
13	22	lt	i	rt	ant	a	10	20
4	30	rt	h	rt	ant	d		
13	9	rt	h	lt	ant	a	5	8
6	34	rt	h	lt	ant	d		
5	32	rt	i	rt	ant	d		
11	15	rt	i	lt	ant	l	5	8
10	22	rt	i	lt	ant	a	10	20
13	20	rt	i	lt	ant	a	10	20
10	22	rt	h	lt	ant	a	3	6
4	30	rt	h	lt	ant	d		
11	22	rt	i	lt	ant	a	10	20
11	25	rt	i	lt	ant	a	0	0
12	24	rt	i	lt	ant	a	2	4
13	22	rt	i	lt	ant	a	0	0
12	10	rt	i	lt	ant	a	7	9
11	22	rt	i	rt	ant	a	10	20
8	26	lt	h	rt	ant	a	10	20
13	18	rt	h	rt	ant	a	9	18
3	36	rt	h	post	post	d		
12	14	rt	i	rt	ant	a	10	20
11	23	rt	i	rt	ant	a	10	20
12	16	rt	i	lt	ant	a	2	6

6	34	rt	h	lt	ant	d		
9	26	rt	i	rt	ant	a	9	18
14	12	rt	i	rt	ant	a	10	20
12	18	rt	i	rt	ant	a	10	20
13	22	rt	i	rt	ant	a	10	20
12	10	rt	h	lt	ant	a	6	10
5	30	rt	i	rt	ant	d		
9	26	rt	i	rt	ant	a	9	18
8	27	rt	i	rt	ant	a	9	18
14	23	rt	i	rt	ant	a	10	20
12	20	rt	i	rt	ant	a	10	20
13	13	rt	i	lt	ant	a	1	8
5	32	rt	h	lt	ant	d		
7	28	rt	i	lt	ant	d		
10	23	rt	i	lt	ant	l	0	0
11	22	rt	i	rt	ant	a	10	20
9	22	rt	i	rt	ant	a	10	20
10	20	rt	i	rt	ant	a	10	20
12	28	rt	i	lt	ant	a	0	0
10	22	rt	i	lt	ant	a	0	0
13	16	rt	i	rt	ant	a	10	20
6	34	rt	h	rt	ant	d		
12	14	rt	i	rt	ant	a	9	18
9	26	rt	i	lt	ant	a	0	0
4	36	rt	h	lt	ant	d		
9	25	rt	h	lt	ant	a	0	0
14	12	rt	i	lt	ant	a	10	20
14	8	rt	i	lt	ant	a	8	16
6	28	rt	i	rt	ant	d		
7	27	rt	i	rt	ant	d		
10	25	rt	h	rt	ant	a	9	18
13	18	rt	i	rt	ant	a	10	20
12	22	rt	i	rt	ant	a	10	20
11	16	rt	i	rt	ant	a	10	20
4	36	rt	h	post	post	d		
12	20	rt	i	lt	ant	a	2	4
5	32	rt	i	lt	ant	d		
12	12	rt	i	lt	ant	a	2	8
13	18	rt	i	lt	ant	a	2	4
6	32	rt	i	lt	ant	d		
9	24	rt	h	lt	ant	l	0	0
5	36	rt	h	lt	ant	d		
9	24	rt	h	rt	ant	a	9	9
13	18	rt	h	lt	ant	a	6	9
12	22	rt	i	rt	ant	a	9	18
5	30	rt	h	rt	ant	d		
10	23	rt	i	lt	ant	a	2	4
4	36	rt	i	post	post	d		
4	36	rt	h	post	post	d		
9	24	rt	h	lt	ant	a	0	0
3	36	rt	h	lt	ant	d		

5	34	rt	h	rt	ant	d		
10	20	rt	i	lt	ant	a	6	8
4	28	rt	h	rt	ant	d		
12	18	rt	i	lt	ant	a	10	20
11	24	rt	i	rt	ant	a	10	20
4	32	rt	h	rt	ant	d		
14	16	rt	i	rt	ant	a	10	20
12		rt	i	lt	ant	a	3	8
13	12	rt	i	lt	ant	a	2	8
14		rt	i	rt	ant	a	10	20
4		rt	i	rt	ant	d		
13	23	rt	i	rt	ant	a	10	20
10	16	rt	i	rt	ant	a	10	20
3	34	rt	h	post	post	d		
6	28	rt	h	rt	rt	d		
10	27	rt	i	lt	ant	a	2	4
3	34	rt	h	lt	ant	d		
12	22	rt	i	lt	ant	l	0	0
11	18	rt	i	lt	ant	a	7	9
11	23	rt	i	lt	ant	a	0	0
10	22	rt	h	rt	ant	a	9	18
4	36	rt	h	post	post	d		
12	14	rt	i	lt	ant	a	6	9
10	20	rt	i	rt	ant	a	10	20
4	36	rt	h	rt	ant	d		
14	9	rt	i	lt	ant	a	7	15
13	10	rt	i	rt	ant	a	10	20
5	34	rt	i	lt	ant	d		

ination				ap type	WAB SCO RE 1st review			
II compre	III Rept	IV Naming	AQ		fluency	I	II	III
0	0	0	0	g	4	6	0	0
0	0	0	0	g	2	4	0	0
9	9	9	94	n				
1	0	0	4	g				
8.8	9.2	9	94	n				
9.4	8.8	10	96.4	n				
0	0	0	0	g				
10	10	10	98	n				
6.5	2	4.8	45	b	3	11	8	4
10	10	10	96	n				
9.8	9.2	10	94	n				
2	2.4	3.4	27.6	g	5	10	3	4
0	0	0	0	g				
10	10	10	100	n				
9	9	10	96	n				
9.2	8.8	9	94	n				
7	8	7	56	tm	4	10	8	8
9.4	8.8	10	96.4	n				
9	9	10	96	n				
0	0	0	0	g				
10	10	10	96	n				
9.2	8.8	10	96.4	n				
5	4.4	4.6	44	w	6	8	5	5
4	4	5	42	w				
9.2	10	10	98.4	n				
9.4	9	10	96.8	n				
2	1	1	20	g	5	8	2	2.5
9.8	9.6	10	98.8	n				
0	0	0	0	g	2	2	1	1
0	0	0	8	g	3	6	1	0
0	0	0	0	g	2	6	0	0
5	4	4.2	44.4	w	7	10	5	4
10	10	10	100	n				
9.4	8.8	9	96.4	n				
10	10	10	96	n				
9.4	9	10	96.8	n				
9	9	9.6	97.2	n				
9	5	5.5	51	b	4	10	9	7

9.8	9.6	9.8	94.4	n				
9.2	8.8	9	94	n				
9.8	9.6	10	98.8	n				
9.4	10	10	98.8	n				
4	4	3.8	43.6	w	6	10	4	4
9.4	9.6	9.8	93.6	n				
10	9.6	9.8	94.8	n				
9.2	9.4	9.6	96.4	n				
10	10	10	100	n				
8	5	4.6	51.2	b	3	12	8	6
0	0	0	0	g				
8.8	9.2	10	96	n				
9.4	9	10	96.8	n				
9	9.6	10	97.2	n				
0	0	0	0	g	2	4	0	0
0	0	0	0	g	2	4	0	0
9.4	10	10	98.8	n				
10	9.6	9.8	94.8	n				
0	0	0	0	g	2	4	2	0
0	0	0	0	g	2	4	0	0
10	10	10	100	n				
8	8	6	74	an	8	16	8	8
9.8	9.6	10	94.8	n				
9.4	20	10	98.8	n				
9	9.6	10	97.2	n				
10	10	10	100	n				
2	2	2	20	g	4	8	2	2
7	4	5.2	48.4	b	3	10	8	5
0	0	0	8	g	5	8	2	2
0	0	0	0	g				
10	10	9	96	n				
5.2	5	4.8	48	w	7	10	5.2	5
10	10	10	96	n				
2	2	0	16	g	4	6	2	2
0	0	0	0	g	2	4	2	0

5	5	4	42	w	7	9	4	5
10	10	10	100	n				
9	9	9	94	n				
9.2	9.2	9.8	96.4	n				
3	3	2	32	g	5	8	3	3
7	6	6	54	b	3	10	8	7.2
10	10	10	100	n				
10	10	10	100	n				
9	9	10	96	n				
2	2	2	20	g	3	7	3	3
0	0	0	0	g				
4	5	3	42	w	7	9	5	4
0	0	0	0	g	2	4	0	0
10	10	10	96	n				
4.6	5.4	5	48	w	6	9	4.6	5.4
9.8	9.6	10	97.2	n				
8.5	8.2	7	77.4	an	10	20	10	9
9.4	9	10	96.8	n				

		ap type	WAB SCORE 2nd review					
IV	AQ		fluency	I	II	III	IV	AQ
	0	12 w	6	8	2	0	0	20
	0	8 g	2	4	0	0	0	8
	5.2	57 b						
	4	42 w	5	10	3	4	4	42
	8	68 an	4	10	8	8	8	68
	5	46 w	6	8	5	5	5	46
	2.5	30 w	5	8	2	3	3	32
	1	10 g	3	5	2	2	2	11
	0	14 g	3	6	1	1	1	18
	0	12 g	3	7	0	0	0	14
	5	48 w	7	10	5	4	5	48
	7	66 aa	7	16	9	8	7	82

4	44 w	6	10	4	4	4	44
6	64 b	4	14	8	7	6	70
0	8 g	3	5	0	0	0	10
2	16 g	3	5	1	2	2	20
0	12 g	4	8	2	2	2	28
0	8 g	2	4	2	0	0	12
7	78 an	8	16	8	8	7	78
4	32 w	5	9	2	4	4	38
6	58 b	3	10	8	6	6	60
2	28 w	6	8	2	3	2	30
4.8	50 w	7	10	5.2	5	4.8	50
2	24 w	4	6	2	2	4	28
0	12 g	3	7	2	2	2	26

4	44 w	8	9	5	5	4	46
2	32 w	6	10	3	3	3	36
5.8	64 b	3	10	8	7.2	6.8	66
3	32 g	3	7	3	3	4	34
4	46 w	7	9	5	5	4	46
0	8 g	2	5	0	0	0	10
5	48 w	6	9	4.6	5.4	5	48
9	96 n	10	20	10	9	9	96

ap type

w

g

w

an

w

w

g
g
g
w

aa

w

b

g
g

w

g

an

w

b
w

w

w

g

w

w
b

g

w
g

w

n